

Definition

Glucosuria, glucose in the urine, results from the glomerular filtration of more glucose than the renal tubule can absorb. It occurs in all normal individuals in amounts up to 25 mg/dl (1–5). Abnormally increased glucosuria [more than 25 mg/dl in random fresh urine (4)], results from either an elevated plasma glucose, an impaired renal glucose absorptive capacity, or both.

The plasma glucose concentration above which significant glucosuria occurs is called the *renal threshold* for glucose. Its value is variable, and deviations occur both above and below the commonly accepted “normal” threshold of 180 mg/dl. In diabetic patients, the value is reported to vary from 54 to 300 mg/dl (6–14). Although glucosuria greater than 25 mg/dl is considered pathologic, many commercial semiquantitative urine tests for glucosuria that are available to patients fail to detect glucosuria until it reaches a level of 50–250 mg/dl (4).

The association between blood and urine glucose was first observed in the eighteenth century by Matthew Dobson, an English physician. For many years urine glucose testing was the major method used to monitor glycemic control in diabetes mellitus. Early methods of urine glucose detection included evaporation of urine to reveal sugar crystals and urine sugar fermentation by yeast. Methods based on copper reduction were developed by von Fehling in the nineteenth century and by Benedict at the turn of the twentieth century. In 1941, the Ames Company marketed Clinistest, a copper reduction method, and followed it with Clinistix, a glucose oxidase-based determination. Since then, several companies have marketed glucose oxidase-based tests.

Measurement of glucosuria is an indirect index of the blood glucose concentration, however, and tests for urine glucose must be interpreted with caution. Technical issues such as test sensitivity and variability of renal glucose threshold must be taken into account. Furthermore, the social stigma sometimes associated with handling a body waste product (15) can be a consideration in terms of patient acceptance of the monitoring technique.

These limitations, together with the development of home blood glucose monitoring, have led to a decline in the use of urine glucose for monitoring in diabetes mellitus. Self-monitoring of capillary blood glucose is now the preferred method. Nevertheless, assessment of glucosuria produces usable data (6,16). It is an especially appropriate monitoring tool in health care settings where socioeconomic or educational constraints (17) preclude the use of more sophisticated techniques (e.g., home capillary blood glucose monitoring, glycosylated hemoglobin measurement, and frequent plasma glucose determinations). Selected patients continue to find urine glucose testing convenient, noninvasive, inexpensive, and useful (18).

Technique

Sample collection for semiquantitative determination of glucosuria in the hospital or the home can take many forms: “24-hour collection” for overall control; “fractional collection” in 4- to 6-hour blocks throughout the day to approximate control points; “first voided spot urine” whose interval is determined by the time since the previous urination; and “second voided spot urine” which assesses glucosuria in a specific short interval since a discarded “first void.” Because of the many factors that alter glucosuria, and because of the time delay between glucose filtration at the glomerulus and its appearance in the void, it is not surprising that several studies indicate that changes in the concentration of glucosuria between the first and second voided urine specimens do not accurately reflect documented change or lack of change in plasma glucose (12,19,20). These observations were substantiated by other studies showing that first and second urine void tests for glucosuria agree only from 62% to 81% of the time (16).

Techniques for measuring glucosuria are based upon either glucose oxidase (specific for glucose) or copper sulfate reduction (nonspecific: detects reducing substances including glucose, fructose, lactose, pentoses, galactose, homogentisic acid, and ascorbic acid). Test-Tape and a variety of “stick” or “strip” tests such as Chemstrip (uG and uGK), Clinistix, Diastix, and Uristix are glucose oxidase based. Clinistest is copper sulfate reduction based. In the glucose oxidase-based technique, hydrogen peroxide is generated and reacts with horseradish peroxidase to produce nascent oxygen. It in turn oxidizes orthotoluidine to produce the blue or purple color that is read. In the Tes-Tape process, oxidized orthotoluidine is reacted with the yellow dye tartrazine to produce a greater range of color development (21).

Multifactorial influences on the presence and degree of glucosuria have produced varying conclusions about how glucosuria should be reported and used in monitoring diabetic control. Confusion is reduced if the absolute value of glucosuria is reported using the “percent scale” now included with all techniques (except Clinistix) as recommended by the ADA Committee on Materials and Therapeutic Agents (22,23).

The sensitivity of commercial clinical “strip” methods can be 10–15 mg/dl (4,7), although 50 mg/dl is usually detected (4). The accuracy of each method also varies with the amount of glucosuria. For example, Clinistest and Diastix agreed with spectrophotometric analysis 78% and 72% of the time, respectively (24). Keto-Diastix and Clinistest were both accurate at lower concentrations; however, Clinistest was correct in 96% of samples known to contain 4% glucose whereas Keto-Diastix was accurate only 40% of the time at this concentration (25). Clinistest is marketed as a two-drop test that

measures glucose concentrations from 0.25 to 5.0 g/dl and as a five-drop test that measures concentrations from <0.25 to 2.0 g/dl. Glucose concentrations above 2.0 g/dl, however, can cause the solution to turn a color similar to that for the 1.0 g/dl reading, the "pass-through" phenomenon. An underestimation of the urine glucose can result (26). Overall, the two-drop copper sulfate procedure (Clinitest) was the most accurate in comparison with spectrophotometric analysis, particularly at lower glucose concentrations (27).

A glucose oxidase-based strip test (Chemstrip uG) has been reported to offer greater accuracy in the clinical range (28). It reportedly excludes upper limit of "normal" values (30 mg/dl) while detecting 60 mg/dl and is comparable to other methods in the 100–250 mg/dl range. On the other hand, Clinitest, Diastix, and Tes-Tape tended to read low between 500 and 2000 mg/dl, whereas Chemstrip uG read high at 2000 mg/dl.

Problems with Glucosuria Measurement Techniques

There is considerable literature about substances that can alter the validity of tests for glucosuria. Conflicting reports often result from the effects of different concentrations of the substance in question or subtle methodologic differences in test procedures (15,16,21,23,28–37). However, achieving a urinary concentration of substances that can alter the test is often difficult under normal clinical circumstances.

In addition to drugs listed in Table 139.1, limited evidence suggests that other agents (nalidixic acid, probenecid, chloral hydrate, hyaluronidase, nitrofurantoin, PAS, phenazopyridine, and iodinated radiopaque agents) can interfere with urine glucose testing (43). Ciprofloxacin, a quinolone antibiotic related to nalidixic acid, does not interfere with Clinitest (44), but has been associated with false positive reactions in certain glucose oxidase tests (45).

Practical issues can also alter test results (46): (a) with Clinitest, angling of the dropper away from vertical can alter volumes that are critical to accurate results (47), (b) timing

and shaking can alter available oxygen and modify the reaction (48), and (c) inattention may cause results to be missed: 58% of nurses routinely performing simultaneous Clinitest and Acetest procedures failed to recognize the "pass-through" phenomenon" (46).

Potential Errors in Monitoring Glucosuria

Errors in color discrimination occur frequently during glucosuria monitoring. Patients of all age groups and professionals read more accurately at the extremes of urine glucose concentrations. Accuracy decreases as the true value approaches the midrange of the test (49). Most errors for all groups are underestimates (50,51). Health care professionals have been trained to balance the error distribution (49), implying that the errors are not inherent to the test procedures.

The acquired color vision deficiency caused by diabetic retinopathy itself may contribute more to inaccuracies in glucosuria testing than any other factor (52). It is primarily a blue–yellow defect and therefore involves most tests for glucosuria. Severity of the diabetes-related defect correlates significantly with overall severity of diabetic retinopathy as well as with the severity of macular disease (52). This effect of diabetes is severe enough to mask the normal loss of hue discrimination which occurs with age. However, hue discrimination can be altered by ambient light as well as by age. Reduced lighting can decrease hue discrimination in people with normal color vision. Although testing in bright lighting did not improve results in nondiabetics, results with diabetic individuals improved significantly (52). Patients with proliferative diabetic retinopathy or diabetic macular disease are probably at higher risk for test errors when monitoring glucosuria.

Errors can also stem from the fallacious concept of a "normal" renal threshold. Real variation in renal thresholds for glucose among individuals is a significant reason why patients and health care professionals find urine tests misleading and are frustrated with the data generated. Further,

Table 139.1
Substances that May Alter Tests for Glucosuria

Substance	Copper sulfate (Clinitest)	Glucose oxidase
Ascorbic acid (4,32,34,38,39)	No effect	False negative
Cephalosporin (30–33,35,40)	False positive	No effect
Monobactam (aztreonam) (41)	High concentrations form a black color; falsely low readings may occur	No effect
Penicillin (35,37)	False positive and false negative	No effect
Levodopa (34,42)	False positive	False negative
Salicylates (34)	False positive	False negative
Nonglucose sugars (4,34)	False positive	No effect
Ketone bodies (15)	No effect	Large amount can repress color development
Uric acid (34)	No effect	False positive
Homogentisic acid (4,34)	False positive	False negative
5-HIAA (21)	False negative	False negative
Peroxide (4,16)	No effect	False positive
Hypochlorite (15)	No effect	False positive
Dilute urine (15,21) (under 100 mOsm/kg)	Falsely high	Falsely high

there is a significant trend for patients with higher renal thresholds for glucose to exhibit higher values for glycosylated hemoglobin and mean blood glucose (6,17): because glucosuria testing cannot warn of hypoglycemia, diabetic patients tend to maintain results at near threshold values. Therefore, in those patients who aim for tight control, glucosuria testing should be abandoned when results are consistently negative. Once management has brought blood glucose to the point of absent glucosuria, there is no alternative to capillary blood glucose monitoring.

Basic Science

Although small amounts of glucose are present in the urine of all normal individuals, the term *glucosuria* is conventionally reserved for pathologic amounts of urine glucose (more than 25 mg/dl in random fresh urine). The renal tubule will reabsorb almost all the glucose present in the normal glomerular filtrate. Glucosuria occurs when that balance is lost: when the amount of glucose in the glomerular filtrate exceeds the capacity of the renal tubule to reabsorb it. The balance can be lost either when the plasma glucose is elevated (e.g., in diabetes mellitus) or when the absorptive capacity of the tubule is impaired (e.g., in Fanconi syndrome, pregnancy, hereditary renal glucosuria, and acute tubular injury).

Renal Threshold for Glucose

There is a negative correlation between the renal glucose threshold and the creatinine clearance in Type I diabetics (7). Age, heart failure, renal disease (e.g., diabetic glomerulosclerosis), and chronic hyperglycemia are known to raise the renal threshold for glucose. Pregnancy, hyperthyroidism, fever, and exercise decrease it (15). In renal disease such as diabetic glomerulosclerosis, a reduced glomerular filtration rate decreases delivery of glucose to the tubule for a given plasma glucose level (53). As a consequence, normal tubular reabsorption of filtered glucose allows the plasma glucose value to rise markedly above the usual threshold before glucosuria occurs (4). Thus, as with age in normal individuals, glomerulosclerosis in long-standing diabetes is associated with a raised renal threshold for glucose, and the presence or amount of glucose in the urine becomes of lesser monitoring value.

Renal thresholds in individual patients may (54) or may not change (6,10) in the short term, but patients with proteinuria have consistently lower renal thresholds (mean 67 mg/dl) (6), and deteriorating renal function in normal individuals tends to result in an increased threshold for glucose (53).

Tubular Reabsorption of Glucose

If the renal tubule capacity for glucose reabsorption is impaired for constitutional or acquired reasons, glucosuria can occur with normal plasma glucose concentrations (4). The Fanconi syndrome, pregnancy, and acute tubular necrosis are examples of this phenomenon. Normally, as the level of plasma glucose and the filtered load rises, renal tubular reabsorption of glucose rises linearly until a maximum tubular resorptive capacity is reached (7). This maximal tubular reabsorptive capacity ranges from 0.9 to 2.0 mmol/

min and is constant for each individual (7). The same is true in diabetes. Patients with recent onset of Type I diabetes exhibited a 20% increase in both glomerular filtration rate and maximal tubular reabsorptive capacity (55). In addition, a reduced glomerular filtration rate in long-term diabetes was accompanied by a lower maximal tubular reabsorptive rate (55). Glomerulo-tubular balance for glucose was maintained in both situations (55).

The proximal convoluted renal tubule reabsorbs most of the filtered glucose load both normally and during hyperglycemia. The intermediate segment, between the late proximal and distal tubule, also can reabsorb glucose (56). It acts as a buffer to aid the response to an increased glucose load; overt glucosuria does not occur until its resorptive capacity is exceeded.

Finally, it has not been possible to demonstrate a correlation between urine flow, maximal tubular reabsorptive capacity, and renal threshold for glucose (7). In addition, the temporal lag between a significant rise in plasma glucose and increased glucosuria varies between 20 and 120 min (6,15). These points deserve special attention when attempting to infer plasma glucose values from results of testing for glucosuria in unstable patients.

Related Pathophysiology

Glucose infusion studies in dogs produced hyperglycemia and raised glomerular filtered load to near maximal tubular reabsorptive capacity without glucosuria (56). Despite resultant reduced fractional proximal reabsorption, and therefore increased glucose egress from the proximal convoluted tubule, the excess glucose load was reabsorbed in the intermediate segment of the renal tubule. The excess filtered load could be shown to inhibit sodium and fluid reabsorption in the proximal tubule. However, sodium and glucose reabsorption could be dissociated and equivalent infusions in which euglycemia was maintained by added insulin had a similar effect, suggesting insulin mediation of the proximal inhibition of sodium and fluid reabsorption.

Rapid infusion of hypertonic glucose elicits different hemodynamic responses in normal and diabetic patients (55). Normal individuals raise renal plasma flow about 10% but exhibit no change in glomerular filtration rate. Diabetics exhibit no change in the renal plasma flow but experience a 9% fall in the glomerular filtration rate. The changes in both groups produce a significant decrease in the filtration fraction, and in both groups there is an increase in urinary sodium excretion during glucose infusion.

Just as the renal threshold for glucose may exhibit wide individual variations, the change in blood glucose necessary to alter glucosuria may vary as well. A group of 65 insulin-dependent diabetic patients studied at home by capillary blood glucose monitoring (6) had a mean renal threshold for glucosuria of 130 mg/dl with a range of 54 to 180 mg/dl. The plasma glucose at which their glucosuria reached 2% varied from 144 to 360 mg/dl, and the plasma glucose change necessary to convert to 2% glucosuria varied from 36 to 288 mg/dl. There was no correlation between individual renal threshold values and the delta plasma glucose necessary to achieve 2% glucosuria.

Methodology used and patient population characteristics appear to influence the factors that can correlate with the renal threshold for glucosuria. Some find that neither age, duration of diabetes, metabolic control, nor the efficiency of tubular glucose reabsorption (splay) are correlated with

the renal glucose threshold (7). Others find a correlation with at least age and mean plasma glucose in patients without proteinuria (14). There is an inverse correlation between maximal tubular reabsorption of glucose and age or duration of diabetes (7). Thus, in a random study of glucosuria in 261 normal persons (5), the mean value for glucosuria in individuals aged 50 years or less was 36.5 mg/dl (95% confidence limits 12.0 to 61.0 mg/dl) and no age- or sex-related differences were observed. However, in individuals over the age of 50 years, the mean values were 25.0 mg/dl (5.0 to 35.0 mg/dl) in men and 14.2 mg/dl (4.0 to 29.0 mg/dl) in women.

Renal glucosuria also occurs. However, the syndrome is rare if one adheres strictly to the criterion of significant amounts of glucosuria after an overnight fast before considering a diagnosis of nondiabetic renal glucosuria. Its primary forms represent a set of related familial genetic defects in which the renal threshold for glucose is altered in two ways: (a) a reduced maximum rate of glucose reabsorption in the tubule; and (b) an altered relationship between the filtered glucose load and glucose reabsorption that does not change maximal reabsorption but does increase the splay of the glucose titration curve. The filtered glucose load at which glucosuria occurs is commonly reduced in both types and distinguishing between them can be difficult. The defects can be either autosomal dominant or autosomal recessive and the conditions usually have no clinical manifestations. Therapy is not required. Their glucose absorption from the gut is normal except for a small subgroup with combined glucose and galactose malabsorption. A patient with complete absence of tubular glucose reabsorption has been reported (57). An additional type of renal glucosuria is reported to occur in patients with nephrotic syndromes (58) and in renal transplant patients recovering from tubular necrosis or acute rejection (59).

Clinical Significance

Measurement of glucosuria is potentially satisfactory for the relatively stable patient with diet and/or oral agent controlled Type II diabetes mellitus who infrequently requires management adjustment, particularly if used in conjunction with periodic assessment of plasma glucose, glycosylated hemoglobin, or fructosamine level. Because of its insensitivity to hypoglycemia, urine glucose testing cannot be generally recommended as a basis for making therapeutic decisions in patients with Type I diabetes or in tightly controlled Type II patients taking insulin. It is also inadequate for use in the management of diabetes in pregnancy (60). However, urine testing in these patients remains necessary for ketones.

Quantitative 24-hour urine glucose determination is advocated by some (61) as a means of assessing glucose control and dietary compliance. A reasonable goal is the excretion of less than 7% of ingested carbohydrate per day. The excretion of much greater amounts in asymptomatic, nonketotic diabetics suggests dietary noncompliance.

Measurement of glucosuria is inexpensive and noninvasive. Patient acceptance of responsibility for monitoring glucosuria can contribute to improved control. In one study (9), the mean blood glucose value was significantly ($p < .02$) lower in patients who complied with glucosuria mon-

itoring instructions (176 ± 81 mg/dl) than in those who did not (200 ± 83 mg/dl).

In patients for whom capillary blood glucose monitoring is impossible or impractical for any reason, we accept and work within the limitations of monitoring glucosuria in order to achieve the benefits which monitoring can offer.

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